1960

Some Effects of Saline Toxicity on Chicks, Laying Hens, Poults, and Ducklings

Laverne M. Krista

SOME EFFECTS OF SALINE TOXICITY ON CHICKS, LAYING HENS, POULTS, AND DUCKLINGS

BY

LAVERNE M. KRISTA

A thesis submitted
in partial fulfillment of the requirements for the
degree of Master of Science at South Dakota
State College of Agriculture
and Mechanic Arts

June 1960

SOME EFFECTS OF SALINE TOXICITY ON CHICKS,

LAYING HENS, POULTS, AND DUCKLINGS

This thesis is approved as a creditable, independent investigation by a candidate for the degree, Master of Science, and acceptable as meeting the thesis requirements for this degree; but without implying that the conclusions reached by the candidate are necessarily the conclusions of the major department.

Thesis Advisor

Head of the Major Department

ACKNOWLEDGEMENTS

The author wishes to express his sincere appreciation to Professors C. W. Carlson, William Kohlmeyer and the entire staff of the Poultry Department for their advice, encouragement and assistance during this work.

LMK

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Some of the waters of South Dakota contain much sodium chloride, sodium sulfate, and magnesium sulfate. Often water from dams, dugouts, and wells will become unsuitable for livestock and poultry consumption because of salt accumulation. Embry, et al., 1959, Bulletin 481, reported that water containing 0.7 percent salt is not satisfactory for livestock.

Salt is an essential ingredient in the poultry ration but relatively small amounts are required. Cases have been reported where chickens suddenly became sick and died. These deaths, which occur without warning, have the characteristics of being caused by microorganisms. Studies conducted on these occurrences revealed that the mortality was due to an excessive intake of salt. Birds may obtain excess salt through the feed or the water.

The primary objective of the experiments reported in this thesis was to determine what level of salt added to Brookings tap water would be toxic to chicks, laying hens, poults, and ducklings.

The mortality, weight gain, and general condition of chicks, poults, and ducklings, and the production and general condition of laying hens were observed.

LITERATURE REVIEW

Some of the early work pertaining to salt toxicity and salt functions was conducted by Zurn, 1882, and Suffram, 1909. Zurn, 1882, reported that 1/2 to 1 ounce of salt will kill a hen in 8 to 12 hours. Suffram, 1909, injected a concentrated salt solution directly into the crop of a chicken after it was fed. It was determined that 4 grams per kilogram body weight of the bird (3 to 5 pounds) was lethal. Observations were made of the birds both ante-mortem and post-mortem. The following ante-mortem observations were reported: hens unable to stand, intense thirst, muscular weakness, convulsions just before death, and viscous discharge from the mouth. Post-mortem examinations showed the following: hemorrhages, severe congestion of the gastro-intestinal tract, and lesions of several organs. Blaxland, 1946, confirmed the work of Suffram, 1909, showing that 4 grams of salt per kilogram of body weight was lethal when injected directly into the crop. When the same level was administered through the feed, excessive thirst and high moisture content of feces were observed but no mortality resulted. Mitchell, et al., 1946; Peterson, 1945; and Quigley and Waite, 1932, also reported that 4 grams of salt per kilogram of body weight were lethal to the bird.

Quigley and Waite, 1932, and Kare and Biely, 1948, have shown that chicks exhibit individual differences within the same age group and at different ages. Mature birds could endure 20 percent salt in the feed;

whereas 5 to 10 percent salt concentrations were very toxic to baby chicks during the first week, Maxland, 1946; Selye, 1943; Quigley, et al., 1932. Mitchell, Card, and Carmen, 1926, reported no detrimental effects with salt in the diet at the 8 percent level for birds 9 to 21 weeks of age. Field, et al., 1946, reported retarded growth at the 8 percent salt level for chicks 9 to 12 weeks of age. They attributed the retarded growth to slow adjustment to the feed, noting that the chicks could tolerate about 6 to 8 grams of salt per bird per day. It was found impossible to induce immediate death by having an excessive amount of salt in the feed. This was primarily due to the lack of palatability of the feed and the additional amounts of water which were consumed with high levels of salt in the diet.

Salt is an essential nutrient in the normal poultry diet, but the level of added salt must be altered according to the amount of sodium chloride contained in the ingredients of the ration for optimum conditions. Prentice, 1933, and Halpin, et al., 1936, reported 0.5 percent to be essential to the young chick for growth, resistance to disease, economical utilization of feed, and early maturity of pullets, and to the laying hen for normal egg production, normal egg size, maintenance of weight, and the prevention of cannibalism and feather pulling. Burns, et al., 1952, conducted a 6 week test and reported that 0.24 percent salt was the lowest level which could be added to the ration and still give satisfactory results in terms of body weight, egg production, and hatchability. A 5 percent added salt level caused a detrimental effect on all of the factors. The main effect of a lack of salt in laying rations was a 20 percent decrease in egg production. Hatchability

was not affected, but if the ration was free of sodium the production dropped to zero. When chlorine was omitted from the diet only a slight change occurred according to Burns, et al., 1952.

The recommendation of the National Research Council for salt in the diet is 0.5 percent. Increased amounts up to a 2 percent level will increase water consumption and raise the moisture content of the feces according to Heuser, 1952, and Halpin, et al., 1936. Peterson, 1945, and Halpin, et al., 1936, suggested that the level of salt should range from 0.5 percent to 1 percent. Slinger, et al., 1950, noted best results in growth rate from salt levels of 0.6 percent. Halpin, et al., 1936, suggested that 1.0 percent salt should be added to the diet because increased growth was obtained at that level. Prentice, 1933, reported equally good results from the use of a 0.5 percent addition of salt to the diet. Owsley and Warto, 1932, and Quigley and White, 1932, found that chicks could tolerate a 30 percent salt ration for a short period. This was far above what would normally be considered the lethal dose. The administration of a 30 percent salt diet fed continuously produced 100 percent mortality, but if the diet was replaced with a normal diet before death occurred the birds made rapid recovery according to Blaxland, 1946.

The amount of sodium required by young chickens is governed by the rate of growth. Sodium and potassium have an interrelationship, one governing the requirement of the other. Sodium and potassium each demonstrate both a sparing and an antagonistic action upon growth, Burns, et al., 1953. The salt requirement of a ration is determined by

the sodium content and not the chloride content, Burns, et al., 1953. Chlorine is required at less than 0.06 percent; therefore the minimum requirement for sodium chloride is equivalent to that for sodium. The National Research Council considers 0.5 percent salt in the diet to be adequate. A level of sodium at 0.16 percent is equivalent to a 0.4 percent sodium chloride diet. A 0.06 percent chloride content is equivalent to a 0.1 percent sodium chloride diet according to Burns, et al., 1953.

An increase in dietary potassium leads to a higher level of sodium in the plasma and an increase in the dietary sodium leads to a lower level of potassium in the plasma, possibly because of increased retention within the cells. This indicates that there may be conditions under which one mineral can promote the retention of the other. The requirement for sodium is about 0.1 percent to 0.3 percent and the requirement for potassium is about 0.23 percent to 0.4 percent, according to Burns, et al., 1953.

Sodium and potassium in the composition of the blood vary as follows: 1. An increase of plasma and whole blood sodium is directly proportional to an increase in the dietary sodium. 2. An increase in dietary sodium at any one level of potassium appears to decrease the level of plasma potassium. 3. At any level of dietary sodium other than 0.2 percent, an increase of the level of potassium in the diet increases the level of plasma sodium, Burns, et al., 1953.

Medway and Kare, 1959, showed that there is a significant difference in the sodium levels of the brain tissue, muscle tissue, and plasma when the diet contains various levels of sodium.

Salt in solution has been reported by Selye, 1943, and Peterson, 1945, to be highly toxic at the 2 percent level, but they observed that salt at the 2 percent level in feed was not detrimental, resulting only in wet litter. Field, et al., 1946, also demonstrated that salt in water was more toxic than the same level of salt administered in the feed. This was explained by Peterson, 1945, in this manner: as the level of salt in the feed was increased, the bird increased its water consumption, thus diluting the salt intake and allowing for survival. However, if the salt is administered in the water the only dilution factor possible is in a limited way by increased feed intake; thus death results.

Toxic effects of salt in the feed began to appear at approximately the 4 percent level according to Kare and Biely, 1948. Paver, et al., 1953, and Krakover, et al., 1948, noted that adverse effects began to appear at and beyond the 3.5 percent and 3.52 percent levels respectively. Paver, et al., 1953, with the 3.25 percent level noted only an increase in water consumption with no effects on feed consumption. In an experiment conducted by Kare and Biely, 1948, the first mortality was recorded at the 4.18 percent level of salt. The chicks on the 4.18 percent level gained and appeared equivalent to chicks on the lower levels. Barlow, et al., 1948, reported a considerable reduction in growth at the 4 percent and 5 percent levels with optimum growth at the 1 percent dietary level for day-old chicks. The birds on the 5.18 percent level had 50 percent mortality, but the survivors showed gains and well-being equivalent to that of chicks on lower levels; whereas, the birds

at the 6.18 percent and 8.18 percent levels, after 29 days on test, gained less and showed an increase in mortality. Individual birds appeared to have an inherent capacity to tolerate or to adapt to high levels of salt.

Pathological abnormalities began to appear when the diet contained 4 percent salt. Individual birds did not show all of the syndromes, but the number and intensity of the syndromes increased with the higher salt concentrations. The syndromes involved at the 4 percent level to the 8 percent level as shown by Kare and Biely, 1948, were as follows: edema ranging from localized to generalized (water in body cavities was common), fluid in the pericardial sac, varying degrees of heart enlargement, gelatinous coverings of the body, gonad enlargement (which proved to be due to liquid accumulation), lung congestion in varying degrees of severity, kidneys swollen or engorged with blood, liver engorged with blood, and ureters filled with chalky material. Physical syndromes noted by Maxland, 1946, at the 5.0, 10.0, and 15.0 percent levels consisted of: heavy mortality, gasping for breath, standing hunched with beak open, closed eyes, staggering gait, spasmodic movement, and generalized edema. On post-mortem examination, an engorgement of the heart, liver, and kidneys were observed.

Kare and Biely, 1948, compared the quantity of salt that was consumed by birds at various levels. They found that chicks on 0.9 percent salt solutions consumed salt approximately equal to that of a 4.13 percent salt concentration in a ration. The water-feed ratio in the two lots was 4.69 cc of water consumed per gram of feed when salt was in

the feed; 4.35 cc of solution were consumed per gram of feed when salt was in the water. A 0.3 percent salt solution was equal to 0.8 percent salt in feed.

The following concentrations of salt solutions were tested on baby chicks by Doll, et al., 1946: 0.25, 0.5, 0.9, 1.5, and 2.0 percent. At the 0.25 percent added salt level the chicks showed no symptoms of salt toxicity. The birds received a standard ration containing 1.0 percent added salt (NaCl). The following are the syndromes which they reported as appearing at the various levels of salt concentrations.

Syndromes and conditions at the 1.5 percent and 2.0 percent salt levels were:

1. Chicks consumed large amounts of water and little feed.
2. The feces had a high water content.
3. Many chicks were sleepy and a few were active.
4. Chicks that were not somnolent had nervous disturbances including opisthotonos, convulsions, incoordination and inability to stand.
5. All chicks died in 4 days; those dying in the first few days were edematous; whereas the last chicks to die were dehydrated.
6. The intestines were slightly pale, edematous, and hemorrhagic.
7. The liver was mottled and yellowish in color.
8. The kidney was slightly enlarged and grayish in color.
9. The heart was dilated and slightly enlarged.
10. The spleen was a pale color.
11. The toxic effects appeared in the first 24 hours; nearly all chicks were affected by the second day.

12. Histological studies revealed:

    a. Albuminous degeneration of heart muscle.

    b. Albuminous degeneration of liver and fatty infiltrations.

    c. Albuminous degeneration of kidney at proximal and distal convoluted tubules.

Syndromes and conditions reported at the 0.9 percent salt level were:

1. A generalized edema and rapid breathing was observed by the third day.

2. Extreme thirst and watery feces occurred by the second day.

3. At the sixth day some chicks were twice the normal weight.

4. Nearly all chicks showed edema at time of death.

5. Edematous conditions gradually disappeared.

6. No variations resulted between groups receiving salt with the feed, or feed without salt.

7. Watery fluid appeared in all parts of the body, peritoneal cavity, pericardial sac, thoracic cavity, and air sacs (few cases).

8. The heart was pale and dilated.

9. The liver was slightly enlarged and pale.

10. The kidney was enlarged and mottled.

11. Fibrin was located in the peritoneal cavity.

12. Chicks increased water consumption and decreased feed consumption.

13. Histological studies revealed:

    a. Albuminous degeneration of heart.

    b. Albuminous, fatty, and hydropic degeneration in hepatic cells.

    c. Desquamation and necrosis of bile duct epithelium.

    d. Albuminous degeneration of proximal and distal convoluted tubules.

e. Nuclear lysis and pycnosis (few cases).

f. Hydropic degeneration in collection tubules.

g. Albuminous degeneration of Bowman's capsule and glomerular epithelium.

h. Interstitial edema present in all sections.

Syndromes and conditions reported at the 0.5 percent salt level were:

1. One-fourth of the chicks developed palpable edema during the first two weeks.

2. The chicks showed symptoms previously described, but conditions developed more slowly.

3. There were no gross lesions at 3 or 4 weeks of age.

4. The growth rate was reduced and the water consumption increased.

5. Histological studies revealed:

a. No change in heart or liver.

b. Albuminous degeneration in Bowman's capsule, the glomerular epithelium and the proximal and distal convoluted tubules.

c. Nuclear pycnosis in some proximal tubules.

d. Collection tubules showed hydropic degeneration and proliferation of interstitial tissue.

A condition in poultry resembling Bright's disease of man has been reported in the northeastern region of the United States and Canada. The terms applied to the disease are numerous: pullet disease, blue comb disease, contagious indigestion, cholera-like disease, summer disease, housing disease, unknown disease, X or XX disease. When the disease appears it will affect the whole flock simultaneously. This condition resembles the action of microbial organisms, but attempts to

transfer the disease have been unsuccessful. While doing experimental work on the pharmacology of corticoid hormones, Selye, in 1943, discovered that an overdose of desoxycorticosterone acetate in the chick elicits nephrosclerosis, cardiovascular changes, water retention, and diarrhea, symptoms which are similar to avian Bright's disease. Soon after this discovery he found that the action of the corticoid hormone is readily obtained in birds receiving 0.3 percent sodium chloride in water. The salt solution greatly increased the sensitivity of the bird to the toxic actions of the desoxycorticosterone acetate, although it causes, by itself, no renal lesions. These results led to the concept that the corticoid hormone acts merely to sensitize the body to the toxic conditions of the sodium chloride and that if a sufficient amount of sodium chloride was administered it would provoke the disease itself.

The toxic conditions are categorized in two ways: the acute (initial or edematous) and the chronic (the edematous condition seems to disappear). In the acute stage the kidney is large, pale, and histologically shows changes, especially in the tubular parts of the nephron. In the chronic stage, the edema tendency gradually disappears; there is a comparative decrease in renal size, and the increasing development of nephrosclerotic changes render the surface of the kidney highly irregular.

In a further experiment, Selye, 1943, placed a group of 4 week old chicks on a 0.3 percent salt solution instead of water. He concluded that there were no clinical nor pathological abnormalities comparable to those elicited by the more concentrated solution.

Selye, 1943, and Krakower and Goettsch, 1945, concluded that poultry were extremely sensitive to small doses of sodium chloride in solution and that 0.9 percent sodium chloride proved quite toxic for fowl and not for mammals. There is a striking resemblance between blue comb disease, Bright's disease, and the experimental sodium chloride toxication. If it were possible that the three disease were one, this would explain why blue comb and avian Bright's disease are not of an infectious origin.

Selye, 1943, administered a 0.9 percent salt solution to chicks to obtain acute and chronic conditions. The syndromes which appeared during the acute stage of the experiment were similar to those listed previously by Doll, et al., 1946, but the detailed conditions pertaining to the kidney itself were not described. The kidney in the acute stage was described as:

> The surface was smooth and in this stage, they resembled the large white kidney of human pathology. Histologically, a marked cloudy swelling of the tubular cells and an increase in the height of the epithelium throughout the nephron, particularly in the proximal and distal convoluted tubules, were observed. Many of the epithelial cells were in the process of desquamation and disintegration. Cellular and even more frequently, hyaline casts were noticeable in many of the tubular lumens. Transudation of fluid was particularly obvious around the adventia of the blood vessels. The glomeruli showed the most characteristic changes, inasmuch as their total size was far above normal due to hyalinization of the capillary wall and marked proliferation of epitheloid cells in and around the capsule of the renal corpuscles. Thus, epithelial cresents were formed, which resembled in every respect those seen in the nephrosclerotic kidney of men.
>
> Between the tenth and the twentieth day of treatment, the edema tendency gradually disappeared, and the birds entered into the second or chronic stage of the disease. Even at this time, there might still be some water accumulation in the pericardium, but most of the other tissues appeared to have lost their water affinity. The heart might still be dilated, but

> in addition to this, signs of cardiac hypertrophy became more and more evident. The tubular changes in the kidneys were somewhat less pronounced than in the acute stage and the stroma edema had practically disappeared, but hyaline casts were still common occurrence and the glomerular changes became increasingly more pronounced with increasing length of treatment. The generalized glomerular sclerosis was accompanied by changes in the medium-sized arteries and veins, inasmuch as during this stage the vessel wall became thicker as the condition progressed. (Ibid.)

Medway and Kare, 1958, began to investigate the concept that the deaths of the animals are caused by dehydration. They proposed the following explanation of the mechanism by which the symptoms and deaths are produced:

> The voluntary ingestion of an excess of salt by an animal or the drenching of an animal with a brine solution is followed by very rapid absorption of salt from the intestine. The rapid absorption of salt results in a marked increase in the osmotic pressure of the blood. This in turn causes a movement of water from the interstitial space into the plasma, initially increasing its volume. At the same time, however, salt diffuses out into the interstitial space. This diffusion of salt into the interstitial space causes a secondary outflow of water from the blood by osmotic action. The net result of this two-way movement is a uniform increase in the NaCl content of the extracellular fluid without change in the relative or absolute water content of plasma or interstitial fluid. The increased NaCl content of the extracellular fluid results in an osmotic pressure greater than that which exists inside the cells. This causes a flow of water out of the cell by osmosis into the extracellular fluid. The final result is uniform increase in the electrolyte concentration and osmotic pressure throughout the body fluids, but there is a decrease in intracellular fluid and an increase in extracellular fluid volume. The moisture determinations of muscle reported here would support such a sequence.

The kidney maintains the osmotic equilibrium of the blood by adjusting the urinary flow. The ingestion of excess salt induces thirst, which causes the consumption of more water, and the water is retained in the system to dilute the ingested salt to restore the osmotic pressure.

The retention of water is due to the antidiuretic hormone (ADH) which is released by the posterior pituitary body. The hypertonicity of the plasma initiates the increase of excessive amounts of ADH.

When an excess of salt is present in the digestive tract and no water is supplied, there will be a massive increase of NaCl throughout the extracellular fluid of the body and a further dehydration of the cells.

In experiments concerning dogs and cats, Medway and Kare, 1958, made observations of cerebrocortical deterioration. They determined that:

> The osmotic pressure of the plasma builds up because of excess salt absorption from the intestine; there is a transitory rise in the cerebrospinal fluid pressure followed by a profound and prolonged drop, recovery occurring under normal circumstances in about 7 hours. This drop in cerebrospinal fluid (CSF) pressure is due to a rapid movement of water out of the CSF into the cerebral blood vessels. As a result of this movement of water out of the CSF the osmotic pressure rises, and this in turn causes an outflow of water from the brain cells.

As these massive shifts of fluid occur, there is a rapid equilibration of NaCl between the plasma and the interstitial cells. When water is present under normal conditions the salt level does not become greatly elevated. Equilibrium occurs rapidly in muscle and other tissue with the exception of the brain and the CSF. This has been substantiated through the use of radioactive tracers. The body seems to have a protective mechanism which protects the nervous system from taking on too many electrolytes and colloids. This is probably the reason for the length of time required to produce nervous symptoms in studies with swine, Medway and Kare, 1958. The death of the animal probably

occurs when the brain barrier is broken down and an inrush of electrolytes occurs.

As the kidney continues to excrete sodium chloride, a hypotonic condition will result between the extracellular fluid of the body and the CSF. This would result in a flow of fluid into the CSF and to the brain. This increases the sodium content of the CSF, and with a lower sodium content in the cells, will result in diffusion of sodium from the extracellular area into the cell. The increase of sodium within the cell will cause an inhibition of glycolysis, and a continuing increase of sodium probably causes the breakdown of the permeability of the cell membrane, with loss of intracellular protein. This would act like a foreign protein and elicit an eosinophilic response. Eosinophilic cuffing of the cerebral blood vessels was evident in the brain of the pig.

Young pigs and chicks are known to be more sensitive to excessive salt intakes than are adult animals. This sensitivity is probably due to their relatively more limited reserves of body water available for salt elimination. The ratio of body weight to surface area is several times greater in the young animal than in the older animal. Since surface area influences the minimal water expenditure, the ratio of minimal water intake to interstitial water volume would be much higher in the young animal. The adult animal reserves are about five times more than the young animal, Kare and Maly, 1965, as quoted by Medway and Kare, 1959.

Medway and Kare, 1959, ran analyses on muscle, brain, whole blood, and plasma for sodium and potassium cations and moisture content. The

cockerels involved were drenched with salt (NaCl) solution. Their results are shown in the following tables.

TABLE I. RESULTS OF ANALYSIS OF COCKEREL MUSCLE, BRAIN, WHOLE BLOOD, AND PLASMA FOR SODIUM AND POTASSIUM (meg/L)

Experiment	No.	Muscle		Brain		Whole Blood		Plasma	
		Na	K	Na	K	Na	K	Na	K
Control	4	16.1	79.1	39.3	79.2	98.3	28.7	147.4	5.0
Feed but no water	3	14.5	78.3	44.6	71.2	94.8	32.0	153.7	5.2
Salt, feed and water	5	18.6	73.1	25.3	77.3	90.3	30.6	150.8	5.7
Salt, feed no water	6	42.6	73.1	75.1	88.4	125.3	34.1	199.0	4.1

TABLE II. MOISTURE DETERMINATION (percent)

Experiment	No.	Muscle	Brain
Control	4	77.0	80.7
Feed but no water	3	75.7	80.5
Salt, feed and water	5	76.7	80.8
Salt, feed but no water	6	73.5	80.7

Salt toxicity of poults was similar to that of chicks. Studies were conducted on salt concentrations in feed and water. According to Scrivner, 1946, conditions occurring with turkeys are ascites, edema,

visceral gout (water belly), and others. The above conditions are characterized by subcutaneous edema, accumulation of fluid in serous cavities, gasping (apparently brought about by pressure on vital organs and edema of lungs), and sudden death.

In the spring of 1949 a new disease was believed to have occurred in poults in Alberta, Canada. It gave negative results to bacterial examination. Higland, 1950, produced similar symptoms of the disease by feeding a 4 percent salt diet. The symptoms which were observed were ascites, hydro-pericardium, edema of lungs and intestinal wall, anaemia of the skeletal muscles, cardiac enlargement, duodenitis, and sudden death with few or no symptoms.

In discussing renal insufficiency MacCallen states:

> The specific inability of the glomeruli to excrete water or, more probably, their specific inability to excrete sodium chloride and other inorganic substances may explain the oliguria or anuria of the acute stage. Sodium chloride passes into the urine through the quite specific activity of some of the epithelial cells, and when these fail, it is retained in the tissue. Since the tissue fluids must remain isotonic for the cells, water is reserved to dilute this concentrated salt solution and the consequence is edema, hydrothorax, and ascites.

Scrivner, 1946, found that salt at the 1 percent level in feed did not produce toxic syndromes in poults. However, salt at the 1 percent level in water resulted in 100 percent mortality within five days. Edema and ascites were evident in all of the latter poults. Salt in solution at the 2 percent level is very toxic. Poults receiving this solution went into a stupor within 48 hours; however, edema and ascites were not evident at the time of death. When salt was at the 2 percent level in the feed, half of the poults on experiment demonstrated

conditions of edema and ascites. James, 1946, concluded that 1 percent salt in the feed was not detrimental to growth or viability, and at the 2 percent level birds increased water consumption and decreased the rate of gain.

It is apparent that sodium even at low levels is the lethal factor. Edema and ascites are produced in poults from sodium compounds and compounds other than sodium. When conditions of edema and ascites occur the primary investigation should be directed toward the sodium intake of the bird.

The conditions occurring most frequently are edema and ascites. Foss, 1941, and Roberts, 1957, conducted experiments with 3 week old poults. The salt in the poult diets ranged from 2 to 8 percent. Poults were affected by salt levels of 4 percent and above. Some symptoms of toxicity appeared at lower levels. Six to eight percent salt levels caused 100 percent mortality during the first week of the experiment. Feed with this level of salt was unpalatable and birds consumed large amounts of water. The levels of salt additions to a diet required to cause edema and ascites will vary with the type of diet being used. This is one reason for the variation of the toxic levels which have been reported by various workers.

Work similar to that of Scrivner, 1946, was reported by Matterson, et al., 1946. He found that poults could tolerate salt up to 2 percent in a semi-purified "salt-free" diet. Batchelder, 1946, reported the same results as Matterson, et al., 1946. Bressler, et al., 1951, fed various levels of salt and found that a 0.9 percent level or

above resulted in a considerable mortality from ascites and visceral edema, but levels at 1.8 percent demonstrated a favorable effect on body weight. The level of salt beginning at the 3.6 percent level was definitely detrimental to body weight.

Atkinson, et al., 1954, conducted an experiment with poults receiving 0.0 percent and 2.0 percent added salt in their diet and determined the carcass moisture content. Ten White Holland poults 25 days of age were placed on experiment. The skin, feathers, and intestinal contents were discarded, and moisture analysis was conducted on the remaining material. Analysis of variance showed that carcasses from poults fed a diet with 2.0 percent added salt were significantly greater in weight and moisture content than carcasses from birds receiving no added salt. The weight differences were entirely due to the moisture content.

Torrey, and Graham, 1935, conducted salt experiments with four six week old Pekin ducks. They determined the lethal dosage of sodium chloride injected into the proventriculus. One and two grams of sodium chloride per day showed no ill effects, but birds receiving four and six grams died the same day and those receiving six grams died within a few hours.

EXPERIMENTAL PROCEDURE

Day-old chicks:

Day-old Single Comb White Leghorn chicks were wing banded, weighed, and randomly distributed in an electrically heated battery brooder. The only known variation between the two experiments was the number of chicks involved in each experiment. The other conditions were essentially the same.

In the first experiment 270 chicks were equally distributed in each of ten pens, and in the second experiment 320 chicks were equally distributed in each of ten pens. In each experiment feed and water (or salt solution) were provided *ad libitum*. The formula for the chick diet, Table III, was the same in each experiment. Duplicate groups of chicks received water containing the following levels of salt 0.0, 0.4, 0.7, 1.0, and 1.2 percent added to Brookings tap water.

TABLE III. CHICK STARTER RATION USED IN BOTH EXPERIMENTS

Ingredients	Lbs.
Ground yellow corn	585.5
Soybean meal	300
Meat scraps	20
Alfalfa meal	20
Dried buttermilk	20
Dicalcium phosphate	20
Fishmeal	20
Yellow grease	5
Salt mix	5
Limestone	2.5
Vitamin A (I.U.)	1800 per lb.
Vitamin D (I.C.U.)	625 per lb.
Riboflavin	2 mg. per lb.
Pantothenic acid	2 mg. per lb.
Niacin	12 mg. per lb.
Choline	52 mg. per lb.
Vitamin B_{12}	4.5 mcg. per lb.
Vitamin E (I.U.)	5 per lb.
Total	998.0

The experiments were conducted for 28 days. Within this period the following records were kept: mortality, body weight, and feed and water (or salt solution) consumption. Each morning the solutions not

consumed were measured out and discarded; the troughs were washed with tap water, and fresh solution was measured into the water troughs.

At the termination of each experiment the control chicks were subdivided into three groups receiving 0.0 percent, 1.0 percent, and 1.2 percent added salt solutions. The chicks in these experiments received the salt solutions and feed ad libitum and were sacrificed after two weeks for tissue and blood analysis. Muscle tissue and liver tissue were analyzed for sodium and potassium content, and the blood plasma was analyzed for sodium content. The flame photometer was used to determine the sodium and potassium levels. The muscle and liver tissues were weighed into samples ranging from 1.2 to 1.8 grams per sample. The samples were oven dried and were then weighed, ashed and then weighed again. The ash was then dissolved in water in a volumetric flask and five milliliters of the solution were analyzed for the sodium and potassium contents. The analysis of the blood plasma was not as complex as that of the muscle and liver tissues. One-half cc of blood plasma was dissolved in water in a volumetric flask, and then five milliliters were analyzed for sodium content.

An analysis of variance was calculated on mortality, weight gain, sodium level, and potassium level. The procedure used was taken from Snedecor, 1957.

Results and Discussion

It is apparent from the data shown in Tables IV, V, and VI, that as the concentrations of salt in water increased, the percent of

mortality increased. The increases in mortality between the control and 0.4 percent groups and the 0.7 percent, 1.0 percent, and 1.2 percent groups were significant. This indicated that the point of toxicity for salt water for baby chicks was in the area of 0.7 percent added salt. The 1.0 percent and 1.2 percent salt levels were more toxic than the 0.7 percent added salt level. Tables IV, V, and VI indicate that during the first week of the experiment mortality increased with the salt concentration. Most of the mortality at the 0.7 percent salt level occurred within the first two weeks of the experiments; whereas for the 1.0 percent and 1.2 percent salt levels most of the mortality occurred during the first week of each experiment.

TABLE IV. MORTALITY OF CHICKS AS INFLUENCED BY ADDITION OF VARIOUS SALT ($NaCl$) LEVELS TO THE DRINKING WATER. (EXPT. I)

% Salt	Pen No.	Initial No.	Termination No.	% Mortality
0.0	I	27	24	11.1
0.0	VI	27	26	3.7
0.4	II	27	25	7.4
0.4	VII	27	26	3.7
0.7	III	27	23	14.8
0.7	VIII	27	18	33.3
1.0	IV	27	5	81.5
1.0	IX	27	4	85.2
1.2	V	27	2	92.6
1.2	X	27	0	100.0

TABLE V. MORTALITY OF CHICKS AS INFLUENCED BY ADDITION OF VARIOUS SALT (NaCl) LEVELS TO THE DRINKING WATER. (EXPT. II)

% Salt	Pen No.	Initial No.	Termination No.	% Mortality
0.0	I	32	31	3.1
0.0	VI	32	29	9.4
0.4	II	32	30	6.2
0.4	VII	31	29	6.5
0.7	III	32	17	46.9
0.7	VIII	32	26	18.7
1.0	IV	32	3	90.6
1.0	IX	32	8	75.0
1.2	V	33	0	100.0
1.2	X	32	2	93.7

TABLE VI. THE INFLUENCE OF ADDED SALT IN DRINKING WATER ON THE TIME OF MORTALITY. (EXPT. I)

Group %	Initial No.	Mortality 1st Wk	2nd Wk	3rd Wk	4th Wk	Total Mortality	Mortality %
0.0	54	4	0	0	0	4	7.4
0.4	54	3	0	0	0	3	5.5
0.7	54	7	5	0	1	13	24.0
1.0	54	29	9	6	1	45	83.3
1.2	54	42	8	2	0	52	96.3

TABLE VII. THE INFLUENCE OF ADDED SALT IN SOLUTION ON THE TIME OF MORTALITY. (EXPT. II)

Group %	Initial No.	Mortality per week				Total Mortality	Mortality %
		1st	2nd	3rd	4th		
0.0	64	4	0	0	0	4	6.2
0.4	63	4	0	0	0	4	6.4
0.7	64	12	6	1	2	21	32.8
1.0	64	36	15	1	1	53	82.5
1.2	65	46	14	2	1	63	96.9

The rate at which mortality, due to salt toxicity, occurred in chicks appeared to be governed by the rate of consumption of the salt in solution, individual tolerances, and adaptability. The ability of some chicks to tolerate various salt levels was shown by the data for weight gain and mortality at the 0.7 percent, 1.0 percent, and 1.2 percent added salt levels.

A reduction in the rate of growth became evident by the second week in each experiment at the 0.7 percent, 1.0 percent, and 1.2 percent added salt levels, Table VIII, and this corresponds with increased consumption of salt (NaCl), Table IX. At the termination of the experiment, the 0.7 percent, 1.0 percent, and 1.2 percent added salt levels had a significant effect on the rate of growth. The initial weights at the beginning of each experiment were relatively similar and the differences between the control and 0.4 percent group and the 0.7 percent, 1.0 percent, and 1.2 percent groups became more

divergent as the experiment progressed. The degree of growth retardation is directly proportional to the salt concentration.

TABLE VIII. THE EFFECT OF ADDED SALT LEVELS ON GROWTH RATE*

Group %	Expt.†	Initial gm	2nd week gm	4th week gm
0.0	I	38.1	125.5	282.9
0.4	I	37.9	125.5	272.5
0.7	I	37.1	119.5	256.1
1.0	I	37.5	91.5	139.8
1.2	I	37.6	67.0	60.5
0.0	II	30.2	90.5	229.2
0.4	II	30.9	95.7	247.4
0.7	II	30.1	87.3	216.7
1.0	II	30.0	67.8	117.5
1.2	II	30.3	59.9	92.5

*These data are the average weights of chicks in duplicate pens.
†Experiment I includes 270 chicks and Experiment II includes 320 chicks.

TABLE IX. THE INFLUENCE OF ADDED SALT LEVELS IN SOLUTION ON THE TOTAL CONSUMPTION OF NaCl (MG).

Group %	Expt.	Gm Feed per Gm Gain	Mg NaCl Intake from Feed*	Ml Sol. per Gm Gain	Mg NaCl Intake from Solution†	Total Mg NaCl per Gm Gain
0.0	I	2.4	18.0	5.2	0	18.0
0.4	I	2.3	17.2	7.1	28.6	45.8
0.7	I	2.1	16.1	10.0	70.3	86.5
1.0	I	3.3	25.1	20.5	205.0	230.1
1.2	I	Inaccurate Data				
0.0	II	2.4	18.0	5.0	0	18.0
0.4	II	1.8	12.6	8.1	32.4	44.8
0.7	II	2.0	15.0	12.3	86.1	101.1
1.0	II	1.7	13.1	19.3	193.0	206.1
1.2	II	2.7	20.6	20.8	249.0	269.6

*The standard ration has 0.5 percent added salt; also the ration contains some fishmeal. Assuming all factors, the ration contains about 0.75 percent salt.

†The calculations are based on the assumption that there is no salt in Brookings tap water.

The intensity and the number of the syndromes became more pronounced as the experiment progressed and the salt concentrations increased. No adverse effects were noted at the 0.4 percent added salt level. Adverse effects of the salt toxicity were evident at the 0.7 percent added salt level but were not as pronounced as with the higher levels of salt.

The most severe effects were noted at the 1.0 percent and 1.2 percent added salt levels. The intensity and the rapidity of the onset of toxicity were proportional to the added salt levels. However, some chicks were able to survive even the highest level of salt given.

The syndromes or conditions which were observed at the various added salt levels, and at the various ages are as follows:

1.0 percent and 1.2 percent added salt levels:

1. Decreased growth.
2. Killing feed and water.
3. Constant chirping and huddling under heating element.
4. Reduced activity and somnolence.
5. Poor feathering (growth and general condition).
6. Labored breathing and mucous discharge from mouth.
7. Nervous actions.
8. Edema.
9. Dehydration.
10. Soft and mushy.
11. Extended wings and drooping head.
12. Watery feces.
13. Increased water (or solution) consumption and decreased feed consumption.
14. Crop filled with liquid.

0.7 percent added salt level:

1. Decreased growth rate.
2. Watery feces.

3. Increased water (or solution) consumption and decreased feed consumption.

4. Slight decrease in activity.

0.4 percent added salt level:

1. Watery feces.

2. Increased water (or solution) consumption and decreased feed consumption.

The mineral analysis conducted on the muscle tissue, liver tissue, and blood plasma showed similar trends in each experiment. The levels of sodium and potassium in the muscle tissue indicated an inverse relationship, Table I, between sodium and potassium. In each experiment the sodium content tended to increase as the salt levels increased while the potassium content decreased. The trends in each experiment were similar, but an analysis of variance indicated that the sodium increase and potassium decrease in the first experiment were significant; whereas the changes in the second experiment were insignificant. There were no significant differences in moisture content or ash content.

TABLE X. THE EFFECT OF ADDED SALT LEVELS IN SOLUTION ON THE SODIUM, POTASSIUM, MOISTURE, AND ASH CONTENT OF MUSCLE TISSUE.

Group %	Expt.	PPM Na	PPM K	Ratio	Percent Moisture	Percent Ash
0.0	I	839.1	3470.0	4.5	72.1	1.31
1.0	I	996.3	3464.1	3.66	71.7	1.32
1.2	I	1231.5	3089.2	2.56	71.8	1.33
0.0	II	981.1	3794.2	3.88	73.7	2.28
1.0	II	1026.9	3727.8	3.63	73.2	2.28
1.2	II	1336.4	3664.5	2.74	72.4	2.47

The analysis conducted on liver tissue indicated no definite changes in sodium or potassium content, Table XI. Also, the moisture and ash values for liver tissue were similar for each experimental group.

TABLE XI. THE EFFECT OF ADDED SALT LEVELS IN SOLUTION ON THE SODIUM, POTASSIUM, MOISTURE, AND ASH CONTENT OF LIVER TISSUE.

Group %	Expt.	PPM Na	PPM K	Ratio	Percent Moisture	Percent Ash
0.0	I	1890.1	2332.4	1.23	72.3	1.38
1.0	I	2040.2	2205.8	1.10	72.1	1.40
1.2	I	1981.1	2399.3	1.23	71.1	1.51
0.0	II	1608.8	2935.7	1.82	73.4	1.86
1.0	II	1524.2	2800.1	1.83	72.7	2.22
1.2	II	1710.9	3324.7	1.94	71.0	1.89

The blood plasma analysed in each experiment showed an increased sodium content with added salt levels as compared to control groups, Table XII. The increase in sodium content is approximately twice as great in the second experiment as in the first. The "analysis of variance" conducted on this data showed that the sodium increase was significant in both experiments.

TABLE XII. THE EFFECT OF ADDED SALT LEVELS IN SOLUTION ON THE SODIUM CONTENT OF BLOOD PLASMA

Group %	Expt.	Sodium PPM
0.0	I	3169.4
1.0	I	3358.0
1.2	I	3645.5
0.0	II	3185.5
1.0	II	3601.8
1.2	II	4059.5

EXPERIMENTAL PROCEDURE

Laying hens:

Two experiments were conducted with 120 Single Comb White Leghorn pullets in each experiment. The pullets were replaced individual cages in a windowless, insulated, and air-conditioned house. The pullets were in the cages for approximately four weeks prior to the beginning of the experiment to permit them to become adapted to their new environment. Each experiment involved the use of eight groups with 15 pullets in each group. In both experiments, three levels of salt were added to the drinking water and administered to duplicate groups.

The first experiment was conducted to determine the effect of sodium chloride additions to the drinking water at levels of 0.4, 0.7, and 1.0 percent. The second experiment was conducted to determine the effects of magnesium sulfate and sodium sulfate in solution. The magnesium sulfate was administered at a 1.0 percent added salt level, and the sodium sulfate was administered at 1.0 percent and 1.2 percent added salt levels. A standard all mash laying ration which contained 0.4 percent added sodium chloride was used in both experiments.

The first experiment was conducted for 16 weeks, and the second experiment was conducted for 12 weeks. During these periods records were kept on the following: mortality, water (or salt solution) consumption, egg production and body weight. Feed records were kept for only the second experiment. Mash feed and water (or salt solution) were provided ad libitum in group troughs. The water troughs were cleaned at intervals of one, two or three days.

TABLE XIII. COMPOSITION OF THE LAYING RATION

Ingredients	Lbs.
Ground yellow corn	371
Soybean meal	50
Meat scraps	25
Alfalfa meal	10
Dried buttermilk	10
Steamed bonemeal	10
Ground limestone	15
Fishmeal	5
Salt mix	2.5
Vitamin A (I.U.)	1800 per lb.
Vitamin D (I.C.U.)	625 per lb.
Riboflavin	2 mg. per lb.
Pantothenic acid	2 mg. per lb.
Niacin	12 mg. per lb.
Choline	52 mg. per lb.
Vitamin B_{12}	4.5 mcg. per lb.
Vitamin E (I.U.)	5 per lb.
Total	496.0

Results and Discussion

Experiment I (NaCl):

In this experiment, feed records were not kept and the water (or salt solution) consumption records were kept only for the duplicate groups, combined. The water (or salt solution) consumption records indicated an increase in water (or salt solution) intake for the 1.0 percent added salt level over the control. The control hens consumed approximately 0.45 pounds of water per hen per day, whereas the hens receiving the 1.0 percent added salt level consumed approximately 1.05 pounds of water per hen per day. The hens receiving 0.4 percent and 0.7 percent added salt levels consumed approximately 0.53 and 0.83 pounds of water per hen per day respectively.

There was a significant drop in egg production at the 1.0 percent added salt (NaCl) level, however, the egg production was not effected at the 0.4 percent and 0.7 percent added salt (NaCl) levels, Figure 1. It is evident that the deleterious effect of the added salt (NaCl) in solution did not affect production until the level of 1.0 percent added salt was reached.

The most noticeable effect of the added salt was the increased moisture content of the feces. The feces became progressively more liquid as the salt concentrations increased. The increase in moisture content became evident at the 0.4 percent added salt level and increased with the concentrations. In general the hens seemed to be unaffected by the salt solutions except for the moisture content of the feces.

The added salt solutions had no significant effect on body weight, mortality, or general condition of the hens. There was little mortality in any of the groups, but the birds that died on the 1.0 percent added salt level had bluish shriveled combs, which is indicative of water starvation. Apparently some of the hens refused to drink the salt solutions at the 1.0 percent added salt level.

Experiment II ($MgSO_4$ and Na_2SO_4):

In this experiment records were kept on water (or salt solution) consumption (individual groups), feed consumption, mortality, body weight, and production. The added salt levels had no significant effect on feed consumption. The control hens consumed 0.32 pounds of water per hen per day whereas the hens receiving solutions of 1.0 percent sodium sulfate, 1.2 percent sodium sulfate, and 1.0 percent magnesium sulfate consumed 0.53, 0.51, and 0.42 pounds of solution per hen per day respectively.

High mortality occurred in one group with the 1.2 percent sodium sulfate level. Eight hens died within three days; the appearance of the hens was indicative of water starvation in that they had bluish shriveled combs. In the other replicate group or the other groups only an occasional death occurred.

The 1.2 percent sodium sulfate and 1.0 percent magnesium sulfate added salt levels reduced egg production significantly, whereas the hens receiving 1.0 percent added sodium sulfate exhibited production records equal to the hens in the control group, Figure 2.

The general appearance of the hens revealed no adverse syndromes traceable to salt toxicity. As in Experiment I, the only noticeable effect due to the added salt levels was the increased moisture content of the feces.

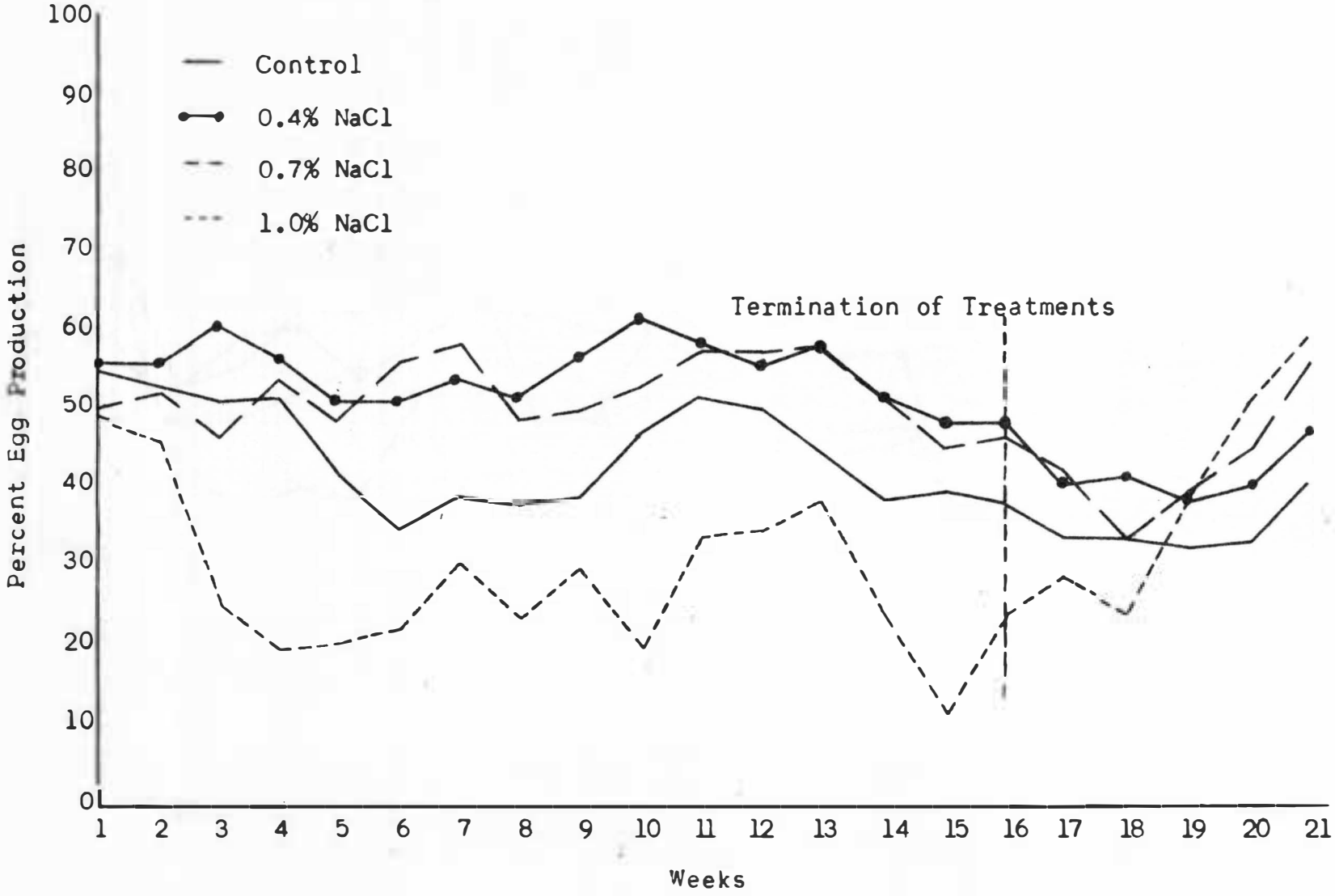

Figure 1. The Effect of Added Salt in Drinking Water on Egg Production.

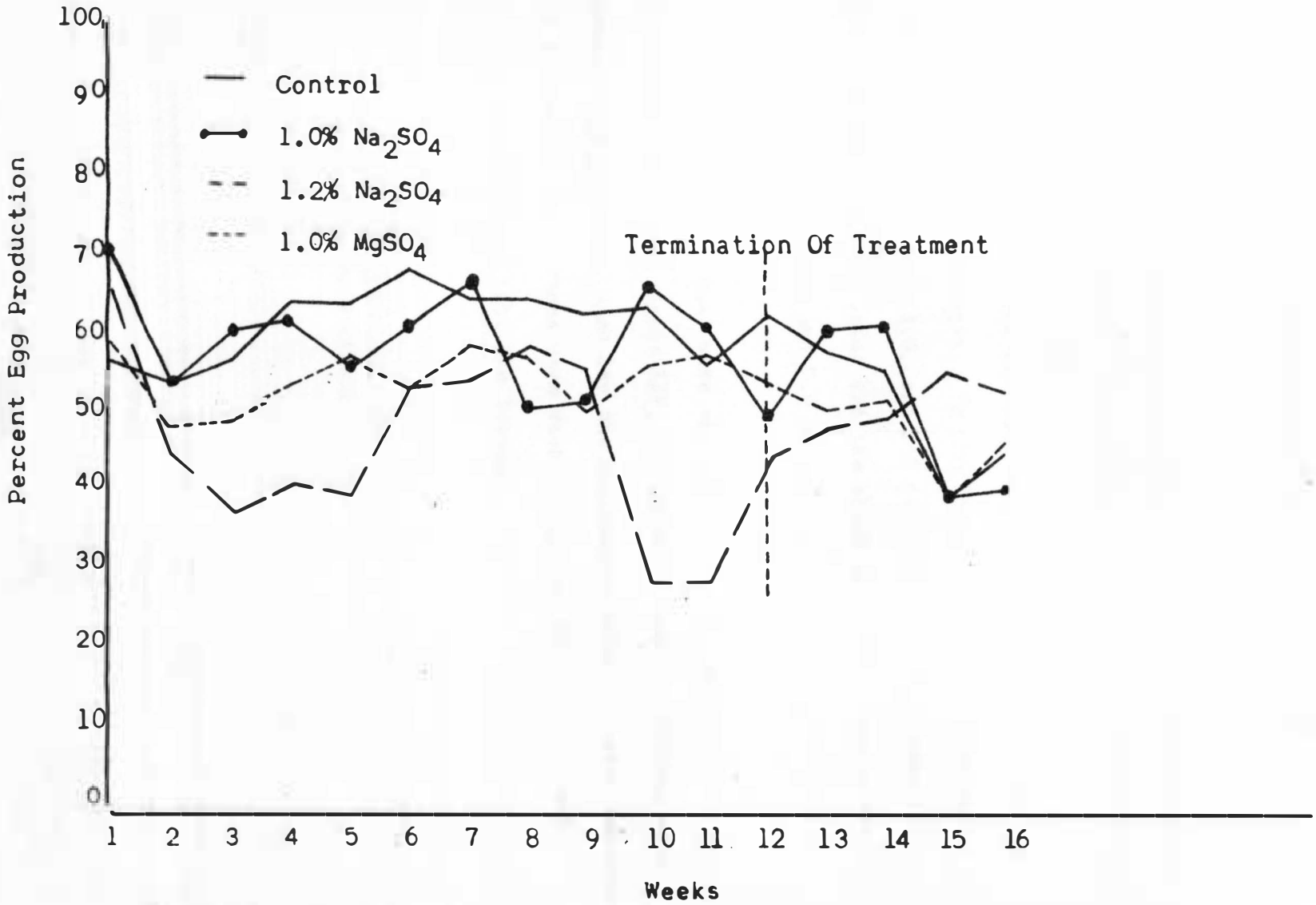

Figure 2. The Effect of Added Salt in Drinking Water on Egg Production.

EXPERIMENTAL PROCEDURE

Turkey poults:

Three experiments were conducted with poults. Day-old poults were wing banded, weighed, and randomly distributed in electrically heated battery brooders. Beltsville White poults were used in the first experiment, and Broad Breasted Bronze poults were used in the second and third experiments. In the first and second experiments levels of 0.0, 0.4, 0.7, 1.0, and 1.2 percent added salt were administered in the drinking water and the third experiment was conducted with levels of 0.0, 0.8, and 0.9 percent added salt using Brookings tap water.

All the experiments were conducted under essentially the same conditions. The feed, Table XIV, and water (or salt solution) were available *ad libitum*. Each day the unconsumed solutions were measured and discarded; the troughs were washed with tap water, and fresh solution was measured into the troughs.

TABLE XIV. TURKEY STARTER RATION USED IN EACH EXPERIMENT.

Ingredients	Lbs.
Ground yellow corn	200
Soybean meal 50%	230
Fishmeal	10
Dried buttermilk	10
Fish soluble conc.	10
Alfalfa meal	10
Limestone	10
Steamed bonemeal	15
Salt mix	2.5
Vitamin A (I.U.)	3600 per lb.
Vitamin D (I.C.U.)	1250 per lb.
Riboflavin	4 mg. per lb.
Pantothenic acid	4 mg. per lb.
Niacin	20 mg. per lb.
Choline	250 mg. per lb.
Vitamin B_{12}	4.5 mcg. per lb.
Vitamin E (I.U.)	5 per lb.
Total	497.5

In the first experiment 245 poults were distributed in ten pens; in the second experiment 95 poults were distributed in ten pens, and in the third experiment 145 poults were distributed in eight pens. The

experiments were conducted for 28 days. During the experiment, records were kept on mortality, weight gain, feed and water (or salt solution) consumption.

Results and Discussion

In the first two experiments a significant increase in mortality occurred beginning with the 0.4 percent added salt level, and the rate of mortality increased with the added salt concentrations, Table XV. In the first two experiments 100 percent mortality was recorded at the 1.0 percent and 1.2 percent added salt levels. The third experiment was conducted with 0.0, 0.6, and 0.9 percent added salt levels. Mortality was 100 percent at the 0.9 percent added salt level and one group at the 0.6 percent added salt level suffered 100 percent mortality; whereas the average mortality of the three groups at the 0.6 percent added salt level was 94.2 percent. The mortality that occurred at the 0.4 percent level occurred during the first week of the experiment; whereas at the 0.7 percent and 0.8 percent levels mortality extended over a three week period. Apparently the poults, at the 0.4 percent added salt level, adapted themselves to the solution. The poults receiving 0.6 percent added salt showed symptoms of toxicity and most of the mortality occurred during the first week of the experiment.

TABLE XV. MORTALITY OF POULTS AS INFLUENCED BY ADDITION OF VARIOUS SALT (NaCl) LEVELS TO THE DRINKING WATER. (EXPT. I)

Group %	Initial No.	Mortality per week 1st	2nd	3rd	4th	Total Mortality	Mortality %
0.0	49	1	1	0	0	2	4.1
0.4	48	15	11	0	0	26	54.1
0.7	49	14	8	4	0	26	53.15
1.0	49	41	8	0	0	49	100.0
1.2	50	49	1	0	0	50	100.0

TABLE XVI. MORTALITY OF POULTS AS INFLUENCED BY ADDITION OF VARIOUS SALT (NaCl) LEVELS TO THE DRINKING WATER. (EXPT. II)

Group %	Initial No.	Mortality per week 1st	2nd	3rd	4th	Total Mortality	Mortality %
0.0	19	1	0	0	0	1	5.0
0.4	19	1	3	0	0	4	20.5
0.7	19	4	7	0	0	11	57.8
1.0	19	16	3	0	0	19	100.0
1.2	19	19	0	0	0	19	100.0

TABLE XVII. MORTALITY OF POULTS AS INFLUENCED BY ADDITION OF VARIOUS SALT (NaCl) LEVELS TO THE DRINKING WATER. (EXPT. III)

Group %	Initial No.	Mortality per week				Total Mortality	Mortality %
		1st	2nd	3rd	4th		
0.0	36	5	3	0	0	8	22.2
0.8	54	34	13	4	0	51	94.4
0.9	54	47	6	1	0	54	100.0

TABLE XVIII. MORTALITY OF POULTS AS INFLUENCED BY ADDITION OF VARIOUS SALT (NaCl) LEVELS TO THE DRINKING WATER. (EXPT. I, II, AND III)

Group %	No. Poults	Mortality per week				Total Mortality	Mortality %
		1st	2nd	3rd	4th		
0.0	104	7	4	0	0	11	10.6
0.4	67	16	14	0	0	30	44.8
0.7	68	18	15	4	0	37	54.4
0.8	54	34	13	4	0	51	94.4
0.9	54	47	6	1	0	54	100.0
1.0	68	57	11	0	0	68	100.0
1.2	69	68	1	0	0	69	100.0

The combined information of the three experiments revealed that the rate of feed consumption decreased with each increase of salt, Table XIX. The rate of water (or solution) consumption increased gradually from the control up to the 0.7 percent added salt level, and above the 0.7 percent added salt level the rate of consumption gradually decreased.

A significant reduction in growth rate occurred at the 0.4 percent added salt level. The reduction in growth rate became evident at 2 weeks of age, Table XIX. The weight reduction paralleled the rate of feed consumption, which was reduced as the salt levels were increased.

TABLE XIX. THE EFFECT OF ADDED SALT LEVELS ON RATE OF CONSUMPTION AND RATE OF GROWTH.

Group %	Average Consumption/Poult/Day*		Average Weights (gm.)*	
	Ml Sol.	Gm Feed	2nd Wk	4th Wk
0.0	57.2	22.9	168.8	460.1
0.4	75.8	21.1	165.5	432.0
0.7	102.6	14.8	139.2	389.7
0.8	58.9	5.7	73.5	266.0
0.9	38.2	2.9	64.0	-----
1.0	35.1	---	----	-----
1.2	24.1	---	----	-----

*Not adjusted for mortality

The syndromes which were observed occurred mainly at the 0.4 percent, 0.7 percent, and 0.8 percent added salt levels. Poults at the 0.9 percent added salt level appeared somnolent, inactive, and consumed little feed. At the 1.0 percent and 1.2 percent added salt levels the poults died before any true syndromes became evident. The poults at the 0.4 percent, 0.7 percent, and 0.8 percent added salt levels showed the following syndromes: retarded growth, inactivity and somnolence, wi t

feces, closed eyes and haunched position, huddling together in groups, and decreased feed consumption.

EXPERIMENTAL PROCEDURE

Ducklings:

Day-old Rouen ducklings were wing banded, weighed, and randomly distributed in an electrically heated battery brooder. The experiment was conducted in two phases. In the first phase of the experiment 24 ducklings were equally distributed in four pens with a control and 0.4 percent, 0.7 percent, and 1.0 percent added salt (NaCl) levels in the drinking water. The second phase of the experiment was performed in conjunction with the first chick experiment. Six ducklings were added to each of the replicate groups of chicks receiving the 1.2 percent added salt levels in the chick experiment (1 or 2).

In each phase of the experiment the feed, Table III, and water (or salt solution) were available *ad libitum*. The environmental conditions and the feed were essentially the same in each phase of the experiment.

The experiment was conducted for 21 days. Within this period records were kept on mortality, feed and water (or salt solution) consumption, and individual body weights at weekly intervals. Each morning the solutions not consumed were measured and discarded; the troughs were washed with tap water, and fresh solution was put into the water troughs.

Results and Discussions

Due to the small number of ducklings involved in this experiment an analysis of variance on the data was not calculated.

Table XX on mortality shows that 100 percent mortality occurred at the 1.2 percent added salt level with 83.0 percent mortality occurring during the first week of the experiment. At the 1.0 percent added salt level 43.0 percent mortality occurred during the second week of the experiment. The 1.0 percent added salt level might be considered as the level producing an increase in mortality.

TABLE XX. MORTALITY OF DUCKLINGS AS INFLUENCED BY THE ADDITION OF VARIOUS SALT ($NaCl$) LEVELS.

Group %	No. Birds	Mortality per week			Total Mortality	Mortality %
		1st	2nd	3rd		
0.0	7	0	0	0	0	0
0.4	7	0	0	0	0	0
0.7	7	0	0	1	1	14
1.0	7	0	3	0	3	43
1.2	12	10	1	1	12	100

The moisture content of the feces and the reduction in body size; Figure 3, were the most prominent conditions induced by the added salt level. The moisture content of the feces increased directly with the increase of the added salt levels while the growth rate was reduced. The ducklings of the control group were almost twice the size of the ducklings at the 1.0 percent added salt level at the termination of the experiment, Figure 3.

Table XXI indicates the reason for the reduction in body size, in that the feed consumption was decreased with increased salt levels. The

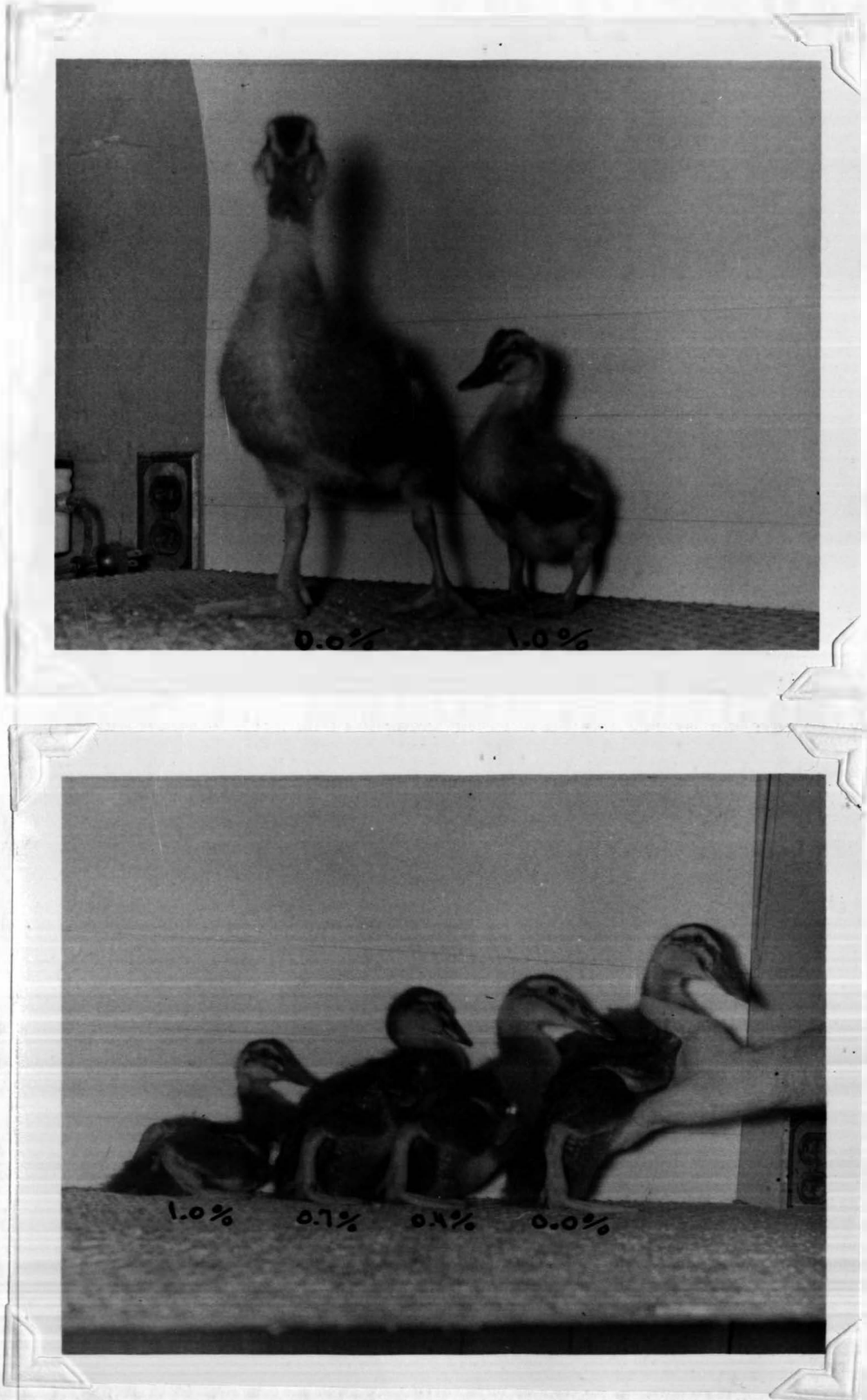

Figure 3. The effect of added salt (NaCl) in tap water on body weight.

highest consumption of water (or salt solution) was at the 0.7 percent added salt level which was 0.96 pounds of solution per day per duckling. The control groups consumed 0.72 pounds of water per day per duckling, and the 0.4 percent and 1.0 percent added salt levels consumed 0.42 and 0.40 pounds of solution per day per duckling respectively.

TABLE XXI. THE EFFECT OF ADDED SALT LEVELS ON RATE OF CONSUMPTION (FEED AND SOLUTION) AND BODY WEIGHT.

Group %	Per Duck Per Day (Ave.)* Ml Sol.	Gm Feed	Initial and Termination Weight Initial	Term.
0.0	325.9	67.17	51.7	687.3
0.4	188.6	53.27	48.9	635.6
0.7	437.8	41.78	50.4	576.8
1.0	179.9	37.06	43.1	356.0
1.2	Inaccurate data			

*Not adjusted for mortality

The general syndromes which occurred in the duckling experiment were most prominent at the 1.0 percent and 1.2 percent added salt levels. Some of the earlier syndromes which occurred at the 1.2 percent added salt level were: retarded growth, wet fronts, billing of feed and solution, decreased feed consumption, and high moisture content of feces. As the experiment progressed, the ducklings exhibited the following syndromes: inactivity, somnolence (eyes closed), bobbing of heads, staggering gait, incoordination, throw head backwards and fall over, rush to watering trough and bill the solution, stagger away and repeat

a cycle similar to the one just performed. When the ducklings reached this stage they soon died.

The conditions with the 1.0 percent added salt level were not as severe; the ducklings which died after receiving this level of added salt did not exhibit the syndromes described for the ducklings on the 1.2 percent added salt level. The syndromes which were evident at the 1.0 percent added salt level were: retarded growth, decreased feed consumption, high moisture content of the feces, billing of feed, inactivity, and standing under the heating element.

The syndromes which were evident at the 0.4 percent and 0.7 percent added salt levels were of the same nature as those described above but not as severe. The conditions which were observed at these levels were as follows: retarded growth, billing of feed and water, and high moisture content of feces. It was quite evident that even the 0.4 percent added salt level had an adverse effect on the ducklings.

SUMMARY

The primary objective of the work conducted with salt water was to determine the level at which deleterious effects could be determined in chicks, laying hens, poults, and ducklings.

Chicks, which were kept in battery brooders, at the 0.4 percent added salt level exhibited well-being equal to that of the control chicks except for a high water content of the feces and increased water consumption. Added salt levels of 0.7 percent, 1.0 percent, and 1.2 percent were deleterious to growth and viability.

Poults are less tolerable to added salt levels in water than chicks. Added salt levels of 0.4 percent were adequate to produce deleterious effects to the poults. A significant increase in mortality and weight reduction occurred at the 0.4 percent added salt level, and these increased with each added salt level.

Mortality and weight reduction occurred at different added salt levels in the duckling experiment. A marked reduction in weight occurred at the 0.4 percent added salt level, and a marked increase in mortality occurred at the 1.0 percent added salt level. Watery feces occurred at each added salt level.

In the experiments with laying hens, sodium chloride, sodium sulfate, and magnesium sulfate salts were used. Watery feces occurred at all salt levels. The added salt levels which appeared to reduce production were 0.7 percent sodium chloride, 1.0 percent sodium chloride, 1.2 percent sodium sulfate, and 1.0 percent magnesium sulfate. The weight

gain and general well-being of the hens were not appreciably affected by the added salt levels.

In each experiment watery feces and increased water consumption occurred at the 0.4 percent added salt levels. The moisture content of the feces and the consumption rate increased proportionally with added salt levels in Brookings tap water.

It is quite evident that salt tolerance will vary with age, individual capacities, and species.

LITERATURE CITED

Atkinson, J. C., R. V. Boucher, and E. W. Callenbach, 1954. Weight and moisture content of carcasses of poults fed added NaCl. Poultry Sci. 33: 656.

Barlow, J. S., S. J. Slinger, and R. P. Zimmer, 1948. The reaction of growing chicks to diets varying in NaCl content. Poultry Sci. 27: 542-552.

Batchelder, 1946. As quoted by Patterson, et al., 1946.

Bigland, C. H., 1950. Ascites and edema of brooded turkey poults in Alberta. Vet. Lab. Dept. Agri. Edmonton, Alberta. Canad. J. Comp. Med. 14: 144-156. Nutritional Abstract 16: 507.

Blaxland, J. D., 1946. The toxicity of NaCl for fowls. Vet. J. 102(6): 157-173. Nutritional Abstract 16: 480

Bressler, G. O., S. Gordeuk, Jr., E. W. Callenbach, and C. K. Pritham, 1951. The effect of NaCl and carbolineum in producing ascites in turkey poults. Poultry Sci. 30: 738-743.

Burns, C. H., W. W. Cravens, and P. H. Phillips, 1952. Requirement of breeding hens for NaCl. Poultry Sci. 31: 302.

Burns, C. H., W. W. Cravens, and P. H. Phillips, 1953. The Na and K requirements of the chick and their relationship. J. Nutrition 50: 317-329. Nutritional Abstract 24: 157.

Doll, E. R., F. E. Hull, and W. M. Insko, Jr., 1946. Toxicity of NaCl solution for baby chicks. Vet. Med. 31: 361-363.

Embry, L. B., H. A. Hoelscher, R. C. Wahlstrom, W. R. Bross, G. F. Gastler, O. E. Olson, C. W. Carlson, and L. M. Krista, 1959. Salinity and livestock water quality. Agri. Exp. Sta., So. Dak. State College, Bul. 481.

Field, H. I., and E. T. R. Evans, 1946. Acute salt poisoning in poultry. Vet. Rec. 58: 253-254. Nutritional Abstract 16: 480.

Forrey, J. P., and R. Graham, 1935. A note on experimental salt poisoning in ducks. Cornell Vet. 25: 50-53. Nutritional Abstract 5: 840.

Foss, J. O., 1941. Salt tolerance of turkey poults. No. Dak. Agr. Expt. St. Mimo. Bul. 4(1):7.

Halpin, J. G., C. N. Holmes, and E. B. Hart, 1936. Salt requirements of poultry. Poultry Sci. 15: 99-103.

Heuser, G. F., 1952. Salt additions to chick rations. Poultry Sci. 31: 85-88.

James, 1946. As quoted by Roberts, R. E., 1957.

Kare, M. R., and H. Biely, 1948. The toxicity of NaCl and its relation to water intake of baby chicks. Poultry Sci. 27: 751-758.

Krakower, C. A., and M. Goettsch, 1945. Effect of NaCl on the chick with particular reference to renal change. Arch. Pathol. 40: 209-219. Nutritional Abstract 19: 399.

MacCallen. As quoted by Scrivner, L. H., 1946.

Matterson, L. D., H. M. Scott, and E. Jungher, 1946. Salt tolerance of turkeys. Poultry Sci. 25: 539-540.

Medway, Wm., and M. Kare, 1959. Water metabolism of the growing domestic fowl with special reference to water balance. Poultry Sci. 38: 631-637.

Mitchell, H. H., E. L. Card, and G. G. Carmen, 1946. The toxicity of salt for chickens. Ill. Agric. Expt. Sta. Bul. 279: 133-156.

Owjley and Warto, 1932. As quoted by Kare and Biely, 1948.

Paver, H., A. Robertson, and J. C. Wilson, 1953. Observations on the toxicity of salt for young chickens. J. Comp. Pathol. 63: 31-47. Nutritional Abstract 23: 699.

Peterson, E. H., 1945. The salt tolerance of chicken. N. Amer. Vet. 26: 37-38.

Prentice, J. H., 1933. The role of salt in poultry nutrition. The J. of the Ministry of Agric. Northern Ireland p. 72. Abst. in International Review of Poultry Sci. 6: 65-66.

Quigley, G. D. and R. H. Waite, 1932. Salt tolerance of baby chicks. Maryland Expt. Sta. Bul. 340: 343-370.

Roberts, R. E., 1957. Salt tolerance of turkeys. Poultry Sci. 36: 672-673.

Scrivner, L. H., 1946. Experimental edema and ascites in poults. J. Amer. Vet. Med. Assoc. 108: 27-32. Nutritional Abstract 16: 481.

Slinger, S. J., W. F. Pepper, and Motzok, 1950. Factors affecting the salt requirements of chicks. Poultry Sci. 29: 780-781.

Selye, H., 1943. Production of nephrosclerosis in the fowl by NaCl. J. Amer. Vet. Med. Assoc. 103: 140-143.

Snedecor, G. W., 1957. Statistical Methods. The Iowa State College Press, Ames, Iowa.

Suffram, 1909. As quoted by Halpin, et al., 1936.

Zurn, 1882. As quoted by Quigley and Waite, 1932.